

Blood in Stool

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GI bleeding can arise from any area of the alimentary tract, and presentation can vary greatly. Bleeding can be insidious with minimal signs or symptoms as with occult blood loss, or dramatic with shock and hematemesis from esophageal varices. Common symptoms include hematemesis (vomiting of blood), the vomiting of “coffee grounds”—appearing emesis, hematochezia (red blood in the stool), and melena (dark black, tarry, foul-smelling stool).

For this discussion we consider only GI sources of bleeding as the cause of blood in the stool, and consider bleeding above the ligament of Treitz as upper gastrointestinal bleeding (UGIB) and below it as lower gastrointestinal bleeding (LGIB). Due to significant variations in presentation, UGIB can be further divided into variceal and nonvariceal bleeding sources. UGIB has an incidence of 40 to 150 episodes per 100,000 population per year, with mortality ranging between 6% and 10%. LGIB accounts for 20 to 27 hospitalizations per 100,000 population per year with mortality rates between 4% and 10%. Additionally, there is a 200-fold increase in the rate of bleeding with advancing age from the third to the ninth decades of life.

Patients with acute, severe, GI bleeding can deteriorate rapidly and immediate and appropriate interventions must be initiated during the primary assessment of the patient. The ABCs—airway, breathing, circulation—of acute resuscitation must be borne in mind at all times.

In addition to bleeding from esophageal varices, nonvariceal bleeding sites including peptic ulcer disease, gastric erosions, Mallory-Weiss tears, and Dieulafoy lesions are discussed. The discussion of LGIB includes diverticulosis, ischemic bowel sources, angiodysplasia, inflammatory bowel disease (IBD), polyps and neoplasia, and anorectal sources.

DIEULAFOY LESION

The characteristic feature is a large-caliber artery in the submucosa, which bleeds on erosion of the overlying mucosa. The lesion can be difficult to locate endoscopically unless actively bleeding. They are usually solitary lesions often in the proximal stomach.

Symptoms

- Dyspepsia ++
- Abdominal or epigastric pain +++
- Hematemesis +++
- “Coffee grounds” emesis +++
- Hematochezia +++
- Melena ++++

Signs

- Hematemesis or “coffee grounds” emesis +++
- Hematochezia +++
- Melena ++++
- Tachycardia and hypotension
- Positive nasogastric aspirate for blood or “coffee grounds” material ++++
- Positive stool testing for blood ++++
- Anemia

Workup

- Evaluate hemodynamic stability immediately. Orthostatic testing: Positive orthostatic testing is variably defined, but usually a drop of at least 20 mm Hg of systolic blood pressure (BP) is considered positive. A report from the American Society of Gastrointestinal Endoscopy (ASGE) showed an improvement in survival if postural changes were not present on initial examination.
- Lab: Hemoglobin, hematocrit, blood urea nitrogen (BUN), creatinine liver function tests, amylase, lipase, and coagulation studies
- In the absence of overt hematemesis, placement of a nasogastric (NG) tube for confirmation of bleeding and assessment of prognosis is generally recommended. NG aspiration has a low false-positive rate, yet may be negative in up to 25% of UGIB.
- Type and crossmatch for 2 to 4 units of packed red blood cells, more if clinically indicated.
- GI consultation for endoscopic evaluation and treatment of the acute bleeding site is recommended. Although most bleeding stops spontaneously, endoscopy provides an accurate diagnosis and the ability to directly coagulate the bleeding site. Various methods of hemostasis are used, but none has been found to be superior to the others.
- Selective visceral angiography should be considered if endoscopy fails to identify a bleeding source. Bleeding must be at rates exceeding 0.5 to 1 mL/min for examination to be optimal and have high yield.
- Radionuclide technetium-99m-labeled red cell scan may also be considered for continued bleeding because it can detect bleeding at rates that exceed 0.1 mL/min. However, pooled blood may sometimes be mistaken for active bleeding.

Comments and Treatment Considerations

Obtain IV access with two large-bore catheters and begin immediate fluid resuscitation with crystalloid solutions as needed. Efforts

to assess the patient's rate and severity of bleeding, and to gather essential historical information to elucidate the possible source of bleeding should occur simultaneously with the evaluation and stabilization of the hemodynamic status.

Assessment is important because a direct correlation has been shown to exist between the number of disease categories present and mortality rates. Risk factors include the use of tobacco, NSAIDs, selective SSRIs, and alcohol, as well as prior *Helicobacter pylori* infection.

In patients awaiting endoscopy, empirical therapy with a high dose PPI should be considered. Omeprazole 8 mg/hr IV for 72 hours is one therapeutic regimen. Long-term acid-suppression therapy with PPIs is recommended along with eradication of *H. pylori* infection using one of the standard treatment protocols.

In certain cases of major bleeding, selective arterial embolization may be attempted, or intra-arterial vasopressin for 24 hours for selective vasoconstriction may be used. This latter therapy has a 70% rate of bleeding control but an 18% rebleeding rate. Indications for surgery include uncontrolled hemorrhage, rebleeding despite endoscopic therapy, large ulceration (>2 cm) or bleeding vessel (>2 mm), continuous posterior duodenal wall bleeding, or transfusion requirement of more than 4 units of blood per 24 hours.



ANGIODYSPLASIA

Angiodysplasia is a submucosal arteriovenous (AV) malformation occurring predominantly in patients between 60 and 80 years old. Their pathogenesis is unknown. They are most commonly found in the proximal colon but can occur anywhere along the GI tract. Bleeding occurs in less than 10% of patients and usually resolves spontaneously, but rebleeding is common.

Symptoms

- Fatigue or weakness +++
- Brisk intermittent rectal bleeding +++
- Syncopal episode

Signs

- Hypotension or tachycardia
- Positive tilt test
- Heme-positive stool +++

Workup

- CBC
- Coagulation studies
- Colonoscopy: 1.5- to 2-mm red patches in the mucosa are seen
- Angiography

Comments and Treatment Considerations

Volume Replacement

Localize bleeding site and stop bleeding with local injection, contact thermal methods, or endoscopic laser therapy. Correct coagulopathy; 80% to 90% of patients will stop bleeding with the preceding measures.

ANORECTAL DISORDERS



ANAL FISSURE

An anal fissure is a tear in the anal mucosa from constipation or trauma. Pain and a small amount of bleeding may occur with bowel movements.

Symptoms

- Anal pain that intensifies during defecation ++++
- Blood on stool surface or on toilet tissue ++

Signs

- Tenderness on digital rectal examination and palpation of fissure (most commonly posterior) +++++
- Visible externally when patient bears down +++

Workup

- Diagnosis is made by the patient's history and gentle physical examination
- Anoscopy may be needed if the fissure is not visualized easily.

Comments and Treatment Considerations

Spontaneous recovery occurs in 60% to 80% of anal fissures. Patients should avoid constipation with a high-fiber diet or the use of a bulking agent such as psyllium. They should take frequent sitz baths and apply topical anesthetic ointment if needed.

A fissure lasting more than 2 months is considered chronic. Nitroglycerin gel (0.2%) daily will heal 50% of chronic fissures. Botulinum toxin injections are an alternative treatment. If medical therapy fails, lateral internal sphincterectomy is the procedure of choice.



FISTULA-IN-ANO

A fistula-in-ano is a tunnel that connects an internal opening, usually at an anal crypt, with an external opening on the perianal skin. Half of patients who undergo incision and drainage of an anorectal abscess will develop a fistula-in-ano.

Symptoms

- Perianal itching ++
- Anal discharge
- Pain

Signs

- Drainage of pus, blood, and sometimes stool from the external opening ++++

Workup

- Anoscopy may reveal the internal opening of the fistula.

Comments and Treatment Considerations

Surgery, fistulotomy, or unroofing of the tunnel are the treatments of choice. Care must be taken to avoid division of the anal sphincter, which could result in fecal incontinence.

**HEMORRHOIDS**

Hemorrhoids are the most common anorectal source of lower GI bleeding. Other sources to consider include anal fissure and fistula-in-ano.

Internal hemorrhoids are located above the dentate line, are covered by mucosa, and more commonly present with bright red bleeding after defecation. Internal hemorrhoids are described by degree of prolapse with grade 1 having no prolapse to grade 4 with a persistent prolapse.

External hemorrhoids are located below the dentate line, are covered with squamous epithelium, have sensory innervation, and when thrombosed present with pain.

Symptoms

- Bright red rectal bleeding on toilet tissue after bowel movement ++++
- Rectal pain usually with bowel movement ++++

Signs

- External hemorrhoids appear as a swollen bluish mass +++++
- Tenderness on rectal examination ++++
- Prolapsed internal hemorrhoids may be visible on examination +++

Workup

- CBC
- Anoscopy can be done without bowel prep in office
- Hemorrhoids are common but other sources of GI bleeding should be investigated with sigmoidoscopy or colonoscopy (especially in patients >50 years).

Comments and Treatment Considerations

Thrombosed external hemorrhoids can be excised within 72 hours of onset. Topical local anesthetics can be used to relieve pain and itching. High-fiber diet and adequate hydration can be used to prevent constipation. Psyllium or docusate can be used to create softer stools. Rubber band ligation of internal hemorrhoids is performed when pharmacologic methods fail. Surgical excision is usually reserved for grade 4 internal hemorrhoids.



COLORECTAL NEOPLASMS

In the United States only lung cancer causes more cancer-related deaths than cancer of the colon and rectum. It is recognized that more than 95% of colorectal cancers develop from benign, neoplastic adenomatous polyps (adenomas). Therefore, it is important to identify and remove adenomatous polyps and early-stage cancers to lower the mortality rate for colorectal cancer. This can be achieved with fecal occult blood testing.

Symptoms

- Abdominal pain (left-sided colon cancer) +++
- Fatigue or weakness (right-sided colon cancer) +++
- Change in bowel pattern or stool caliber/tenesmus (left-sided colon cancer) +++
- Weight loss +++

Signs

- Heme-positive stool ++++
- Mass on rectal examination
- Tenderness on abdominal palpation

Workup

- CBC
- Coagulation studies
- Liver function tests
- Colonoscopy with biopsy
- Computed axial tomography (CAT) scan of the abdomen and pelvis

Comments and Treatment Considerations

All patients with colorectal cancer should be staged because prognosis is closely associated with depth of tumor penetration, presence of regional lymph nodes and distant metastasis. The most frequently used staging system is the Dukes classification.

Resection offers the greatest potential for cure in patients with invasive colorectal cancer. Before surgery, a carcinogenic embryonic antigen (CEA) titer should be determined and colonoscopy performed. CEA assay is not useful as a screening test because elevations usually occur with cancers of at least stage B2.



DIVERTICULAR DISEASE OF THE COLON

Colonic diverticuli are herniations of colonic mucosa and submucosa through the muscularis propria. Most diverticular disease in Western countries occurs in the left colon, although 50% of diverticular bleeding originates from a diverticulum proximal to the splenic flexure. Diverticulitis results from inflammation and/or perforation of a diverticulum. Diverticulosis is more common in women and whites and generally presents with pain and not GI bleeding. Only 10% to 25% of patients with diverticulosis will develop diverticulitis.

Symptoms

- Abdominal pain especially in the left lower quadrant ++++
- Change in bowel habit ++
- Nausea or vomiting ++
- Orthostatic symptoms if acute blood loss or chronic over a long period of time +

Signs

- Elevated temperature (usually $<38.5^{\circ}\text{C}$ but may be higher) ++++
- Tenderness and/or mass palpated in the left lower quadrant ++++
- Heme-positive stool++
- Tachycardia or hypotension (occurring with complications such as abscess, fistula, or perforation)

Workup

- CBC
- ESR or CRP
- Coagulation studies
- Type and crossmatch if hemodynamically unstable.
- Helical CT of the abdomen with oral water-soluble contrast
- Bleeding scan with technetium helps localize the site of bleeding when rate is greater than 0.5 mL/min
- Angiography

Comments and Treatment Considerations

A stable patient who can maintain oral fluids and has mild diverticulitis may be managed as an outpatient with a liquid diet and oral antibiotics to cover gram-negative and anaerobic bacteria. Patients with moderate to severe diverticular disease should be managed as an inpatient. Management includes keeping the patient NPO, volume replacement with IV fluids and /or blood products, Foley catheter to monitor output, NG tube insertion, and pain management.

Surgical intervention may be needed for perforation, abscess/fistula formation, stricture or obstruction. Long-term medical management for patients with diverticular disease includes high-fiber diet and avoidance of food with seeds, nuts, and kernels.



INFLAMMATORY BOWEL DISEASE

Ulcerative colitis and Crohn's disease are the two most common inflammatory bowel disorders although their etiologies still remain unknown. These diseases are most common in the second and third decades of life, affecting males and females equally. A genetic predisposition exists and cigarette smoking may affect the disease.

Symptoms

- Blood in stool (gross or hematochezia) ++++
- Crampy abdominal pain ++++
- Diarrhea (more often bloody in ulcerative colitis) ++++
- Urgency to have a bowel movement ++++

Signs

- Elevated temperature ++++
- Weight loss ++++
- Heme-positive stools (ulcerative colitis) +++
- Hypotension/tachycardia
- Diffuse abdominal tenderness that may have associated rebound, guarding, or rigidity

Workup

- CBC
- Electrolytes
- ESR or CRP
- Serum albumin
- Stool studies such as WBC, ova, and parasites
- Colonoscopy with biopsy
- CT of abdomen with oral contrast

Comments and Treatment Considerations

Medical

- Aminosalicylates
- Steroids
- 6-Mercaptopurine
- Antirheumatic drugs such as azathioprine, methotrexate, and infliximab

Surgery

Indications for surgery include failure to respond to medication, cancerous or precancerous changes, fistula, abscess, or stricture.



LOWER GASTROINTESTINAL BLEEDING

Lower GI bleeding when acute causes frequent hospital admissions and affects morbidity and mortality. Most series indicate the average age of patients with lower GI bleeding is 60 years old. The most common causes of lower GI bleeding are diverticular disease of the colon, IBD, anorectal disorders, neoplasms, and angiodysplasias.

MALLORY-WEISS TEAR

A Mallory-Weiss tear is a longitudinal laceration of the mucosa usually near the gastroesophageal junction caused by forceful retching or vomiting.

Symptoms

- Forceful retching or vomiting precedes hematemesis +++
- Small volume of blood in vomitus ++++

Signs

- Hematemesis +++
- Hematochezia and/or melena
- Tachycardia and hypotension
- Positive nasogastric aspirate for blood +++
- Positive stool testing for blood
- Anemia

Workup

- See workup for PUD or gastric erosions.

Comments and Treatment Considerations

The history of antecedent forceful retching is key to making this diagnosis. Bleeding is usually minimal and self-limited. Treatment should focus on symptom control and treatment of nausea or the underlying cause of the vomiting. If there are symptoms of GERD, PPI therapy should be initiated. For continuing bleeding or hemodynamic instability, treatment is as noted under "Peptic Ulcer Disease/Gastric Erosions."

PEPTIC ULCER DISEASE/GASTRIC EROSIONS

PUD, gastric erosions, a Mallory-Weiss tear, and the Dieulafoy lesions are all examples of nonvariceal bleeding, and represent more than two thirds of the sources of major UGIB. PUD and gastric erosions have similar risk factors, and are often associated with heavy alcohol use and the use of NSAIDs.

Scoring systems have been developed in an attempt to identify patients at high risk for continued bleeding or rebleeding. However, the difficulty in using these systems is that an important question remains unanswered: whether endoscopy is essential or whether some population of patients can be detected clinically who do not require endoscopy.

Assessment for comorbid conditions is important because the presence of a higher number of comorbid conditions is associated with poorer prognosis. Other factors that increase the risk of morbidity and mortality are advancing age, hemodynamic instability, and increased bleeding intensity.

A peptic ulcer is a localized process resulting from the erosion of the gastric mucosa exposing the submucosa. Gastric erosion (gastritis) is a more generalized process encompassing a larger

surface area of the gastric mucosa and is accompanied by diffuse inflammatory changes. Bleeding is the result of erosion extending to the underlying blood vessel, and the rate and severity of bleeding are determined by the size of the vessel(s) involved.

Symptoms

- Dyspepsia ++
- Abdominal or epigastric pain +++
- Hematemesis +++
- “Coffee grounds” emesis +++
- Hematochezia +++
- Melena ++++

Signs

- Hematemesis or “coffee grounds” emesis +++
- Hematochezia +++
- Melena ++++
- Tachycardia and hypotension
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- Positive stool testing for blood ++++
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Workup

- Evaluate hemodynamic stability immediately. Orthostatic testing: Positive orthostatic testing is variably defined, but usually a drop of at least 20 mm Hg of systolic BP is considered positive. A report from the ASGE showed an improvement in survival if postural changes were not present on initial examination.
- Lab: Hemoglobin, hematocrit, BUN, creatinine (BUN:creatinine ratio of >36:1 in patients without renal failure is suggestive of UGIB), liver function tests, amylase, lipase, and coagulation studies
- In the absence of overt hematemesis, placement of an NG tube for confirmation of bleeding and assessment of prognosis should occur. NG aspiration has a low false-positive rate yet may be negative in up to 25% of UGIB.
- Type and crossmatch for 2 to 4 units of packed red blood cells, more if clinically indicated.
- GI consultation for endoscopic evaluation and treatment of the acute bleeding site is recommended. Although most bleeding stops spontaneously, endoscopy provides an accurate diagnosis and the ability to directly coagulate the bleeding site. Various methods of hemostasis are used, but none has been found to be superior to the others.
- Selective visceral angiography should be considered if endoscopy fails to identify a bleeding source. Bleeding must be at rates exceeding 0.5 to 1 mL/min for examination to be optimal and have high yield.
- Radionuclide technetium-99m-labeled red cell scan may also be considered for continued bleeding because it can detect bleeding at rates that exceed 0.1 mL/min. However, pooled blood may sometimes be mistaken for active bleeding.

Comments and Treatment Considerations

Obtain IV access with two large-bore catheters and begin immediate fluid resuscitation with crystalloid solutions as needed. Efforts to assess the patient's rate and severity of bleeding, and to gather essential historical information to elucidate the possible source of bleeding should occur simultaneously with the evaluation and stabilization of the hemodynamic status.

Assessment regarding prior UGIB and the presence of comorbid conditions are important because a direct correlation has been shown between the number of disease categories present and mortality rates. Risk factors include the use of tobacco, NSAIDs, SSRIs, and alcohol, as well as prior *H. pylori* infection.

In patients awaiting endoscopy, empirical therapy with a high-dose PPI should be considered. Omeprazole 8 mg/hr IV for 72 hours is one therapeutic regimen. Long-term acid-suppression therapy with PPIs is recommended for PUD, along with eradication of *H. pylori* infection using one of the standard treatment protocols.

In certain cases of major bleeding from PUD and gastritis, selective arterial embolization may be attempted, or intra-arterial vasopressin for 24 hours for selective vasoconstriction may be used. This latter therapy has a 70% rate of bleeding control but an 18% rebleeding rate. Indications for surgery include uncontrolled hemorrhage, rebleeding despite endoscopic therapy, large ulceration (>2 cm) or bleeding vessel (>2 mm), continuous posterior duodenal wall bleeding, or transfusion requirement of more than 4 units of blood per 24 hours.

Although EGD is the gold standard for evaluation of UGIB, exactly when it should occur in the hemodynamically stable patient is not established. Most studies refer to EGD within 24 hours of admission.

UPPER GASTROINTESTINAL BLEEDING

UGIB is five times more common than LGIB, and often presents with hematemesis or “coffee grounds” emesis, but can also present with hematochezia or melena. The initial assessment for UGIB should begin with a rapid assessment of the patient's hemodynamic stability. Rapid transfer to an inpatient setting is appropriate when any hemodynamic instability is found.

VARICEAL BLEEDING

Esophageal varices are abnormally dilated veins found predominantly in the distal esophagus, are often associated with cirrhosis, and are the result of increased portal pressure. They are the most common single cause of “severe and persistent” UGIB (33% of cases), are significantly more likely to present with bright red hematemesis (76% versus 49% for ulcers), and have a mortality rate of about 30% or greater (Table 10-1).

Table 10-1. Causes of Major Upper Gastrointestinal Bleeding

DISEASE STATE	% OF BLEEDING
Peptic ulcer disease	60%
Variceal bleeding	10%-30%
Gastric erosions	6%-30%
No diagnosis	7.6%-22%
Mallory-Weiss tear	5%-11%
Dieulafoy lesion	5%

Symptoms

- Sudden onset of vomiting a large volume of bright red blood with or without clots ++++

Signs

- Tachycardia
- Positive orthostatic testing
- Hemodynamic instability
- Anemia
- Hematochezia

Workup

- Initial evaluation of hemodynamic stability. Orthostatic testing: Positive orthostatic testing is variably defined, but usually a drop of at least 20 mm Hg of systolic BP is considered positive. One report showed an improvement in survival if postural changes were not present on initial examination.
- Lab: hemoglobin, hematocrit, BUN, creatinine (BUN:creatinine ratio of >36:1 in patients without renal failure is suggestive of UGIB), liver function tests, amylase, lipase, and clotting studies
- Type and crossmatch for 2 to 4 units of packed red blood cells
- Depending on age, ECG and cardiac monitoring may be appropriate.
- Emergent GI consult and endoscopy are required for endoscopic treatment with sclerotherapy or band ligation.

Comments and Treatment Considerations

Obtain IV access with two large-bore catheters and begin immediate fluid resuscitation with crystalloid solutions as needed. Assess the vital signs quickly and repeatedly for hypotension or postural hypotension. Intensive care unit (ICU) admission is indicated for hemodynamically unstable patients.

Consider octreotide 25 to 50 mcg/hr IV infusion for 5 days in combination with endoscopic therapy (works better than either therapy alone). Consider vasopressin drip at 0.2 to 0.4 unit/min IV (max is 0.9 unit/min), taper after 12 hours. Give with nitroglycerin (NTG) IV; this regimen has a 50% success rate, and a high rebleeding

rate. For continued bleeding despite therapy, consider intubation and insertion of a Sengstaken-Blakemore tube pending surgical/radiologic evaluation.

If bleeding persists, consider the insertion of a transjugular intrahepatic portosystemic shunt (TIPS) by an interventional radiologist. Although TIPS has many contraindications, it is also reported to control bleeding in 90% of patients. However, this procedure has a high rate of complications and a mortality of 1% to 2%. Surgery is considered when other measures have been ineffective. Procedures include portosystemic venous shunting and esophageal devascularization.

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